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L3 ANSWER 1 OF 14 BIOSIS COPYRIGHT 2002 BIOSIS
2001:285500 Document No.: PREV200100285500. Pharmacologic intervention in the fibrotic response. Martin, George Reilly (1). (1) FibroGen, Inc., San Francisco, CA USA. IOVS, (March 15, 2001) Vol. 42, No. 4, pp. S1. print. Meeting Info.: Annual Meeting of the Association for Research in Vision and Ophthalmology Fort Lauderdale, Florida, USA April 29-May 04, 2001

Language: English. Summary Language: English.

L3 ANSWER 2 OF 14 BIOSIS COPYRIGHT 2002 BIOSIS
2001:314023 Document No.: PREV200100314023. Gene induction by coagulation factor Xa is mediated by activation of protease activated receptor-1. Riewald, Matthias (1); Kravchenko, Vladimir (1); Petrovan, Ramona J. (1); Brass, Lawrence F.; Ruf, Wolfram (1). (1) Dep of Immunology, The Scripps Research Institute, La Jolla, CA USA. Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 447a. print. Meeting Info.: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000 American Society of Hematology. ISSN: 0006-4971. Language: English. Summary Language: English.
AB In addition to activating prothrombin, the coagulation protease factor Xa (Xa) triggers a variety of cellular responses, including induction of inflammatory genes. We found that Xa and thrombin, but not other coagulation serine proteases, induced nuclear factor-kB (NF-kB) in a HeLa cell line that expresses protease activated receptor (PAR)-1, but not PAR-2, -3, and -4. Xa in the presence of the specific thrombin inhibitor hirudin induced NF-kB in HeLa cells efficiently, but with delayed kinetic compared to thrombin. This delay caused no difference in gene expression patterns, as determined by high-density microarray analysis. Both proteases most prominently induced the angiogenesis promoting genes Cyr61 and **connective tissue growth factor** (CTGF) on the panel represented by the UniGEM V chip (Incyte Corp.). Inhibition of PAR-1 cleavage with **monoclonal antibodies** abolished MAP kinase phosphorylation and gene induction by Xa, demonstrating that Xa signals through PAR-1 and not through a novel member of the PAR family. The snake venom prothrombin-activating enzyme Ecarin also produced PAR-1 dependent signaling that was completely blocked by the thrombin inhibitor hirudin, demonstrating that prothrombin remained cell associated during serum starvation at levels that are sufficient to allow for PAR-1 activation. The efficacy of hirudin to block in situ generated thrombin excludes thrombin as an intermediate in Xa-dependent PAR-1 activation which was not inhibited by hirudin. The concentration dependence of Xa for PAR-1 activation is consistent with previously characterized Xa-mediated PAR-2 signaling, suggesting that local concentration of Xa on the cell surface, rather than sequence specific recognition of the PAR scissile bond is determining receptor cleavage. This study demonstrates that PAR-1 cleavage by Xa can elicit the same cellular response as thrombin, but mechanistic differences in receptor recognition may be crucial for specific roles for Xa in signaling in spatial or temporal separation from thrombin generation.

L3 ANSWER 3 OF 14 MEDLINE DUPLICATE 1
2000492323 Document Number: 20365924. PubMed ID: 10906042. Insulin-like growth factor (IGF)-binding protein-4 proteolytic degradation in bovine, equine, and porcine preovulatory follicles: regulation by IGFs and heparin-binding domain-containing peptides. Mazerbourg S; Zapf J; Bar R S; Brigstock D R; Monget P. (Station INRA de Physiologie de la Reproduction des Mammiferes Domestiques, URA CNRS 1291, 37380 Nouzilly, France.) BIOLOGY OF REPRODUCTION, (2000 Aug) 63 (2) 390-400. Journal code: A3W; 0207224. ISSN: 0006-3363. Pub. country: United States. Language: English.
AB We recently showed that insulin-like growth factor-binding protein-4 (IGFBP-4) proteolytic degradation in ovine preovulatory ovarian follicles is IGF-dependent and regulated by the heparin-binding domain (HBD) from IGFBP-3 and from **connective tissue growth factor** (CTGF), heparan/heparin-interacting protein (HIP), and vitronectin. The present study investigated regulation of IGFBP-4 proteolytic degradation in porcine, bovine, and equine ovarian preovulatory follicles. Follicular fluid from such preovulatory follicles contains proteolytic activity, degrading exogenous IGFBP-4. An excess of

IGF-I enhanced IGFBP-4 degradation. In contrast, IGFBP-2 or -3 or **monoclonal antibodies** against IGF-I or -II dose-dependently inhibited IGFBP-4 degradation, and IGF-I or -II reversed this inhibition in a dose-dependent manner. Heparin-binding peptides derived from the C-terminal domain of IGFBP-3 or -5 inhibited IGFBP-4 degradation. Other heparin-binding peptides derived from CTGF, HIP, and vitronectin also inhibited IGFBP-4 degradation, except in porcine follicles. Finally, IGFBP-3 that was mutated in its HBD was less effective at inhibiting IGFBP-4 degradation. Thus, in bovine, porcine, and equine preovulatory follicles, IGFBP-4 proteolytic degradation both depends on IGFs and is inhibited by peptides containing HBD. Overall, these results suggest that during terminal development of follicles to the preovulatory stage in domestic animal species, the increase in IGF bioavailability might enhance IGFBP-4 degradation. In contrast, in atretic follicles, the decrease in IGF bioavailability, resulting partly from the increase in IGFBP-2 (sow, heifer, mare) and IGFBP-5 (heifer) expression would participate in the decrease of IGFBP-4 degradation. In bovine atretic follicles, IGFBP-5 would also strengthen the inhibition of IGFBP-4 degradation by direct interaction of its HBD with the protease. The involvement of other HBD-containing proteins in the modulation of intrafollicular proteases degrading IGFBP-4 remains to be investigated.

L3 ANSWER 4 OF 14 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

2000040274 EMBASE Serum levels of **connective tissue growth factor** are elevated in patients with systemic sclerosis: Association with extent of skin sclerosis and severity of pulmonary fibrosis. Sato S.; Nagaoka T.; Hasegawa M.; Tamatani T.; Nakanishi T.; Takigawa M.; Takehara K.. Dr. S. Sato, Department of Dermatology, Kanazawa Univ. School of Medicine, 13-1 Takaramachi, Kanazawa, Ishikawa 920-8641, Japan. s-sato@med.kanazawa-u.ac.jp. Journal of Rheumatology 27/1 (149-154) 2000.

Refs: 33.

ISSN: 0315-162X. CODEN: JRHUA. Pub. Country: Canada. Language: English. Summary Language: English.

AB Objective. To determine the serum levels and clinical correlation of **connective tissue growth factors** (CTGF) in patients with systemic sclerosis (SSc). Methods. Serum samples from patients with limited cutaneous SSc (lSSc, n = 32), diffuse cutaneous SSc (dSSc, n = 28), systemic lupus erythematosus (SLE, n = 30), polymyositis/dermatomyositis (PM/DM, n = 20), and healthy control subjects (n = 30) were examined by ELISA for detection of CTGF. Results. Serum CTGF levels in patients with SSc were significantly higher than those in patients with SLE or PM/DM, and in controls. CTGF levels in patients with dSSc were significantly higher than those in patients with lSSc. As for clinical correlation of CTGF, SSc patients with elevated CTGF had pulmonary fibrosis, decreased DLCO, and decreased vital capacity more frequently than those with normal CTGF levels. Further, DLCO and vital capacity were inversely and directly correlated with serum CTGF levels in patients with SSc. The dSSc patients with disease duration of 1-3 years had significantly elevated levels of CTGF compared with dSSc patients with duration < 1 year or more than 3 years. Conclusion. Serum CTGF levels were increased in patients with SSc, and correlated with the extent of skin sclerosis and the severity of pulmonary fibrosis. In addition, it appears that production of CTGF is involved in the development or maintenance of fibrosis rather than in initiation of fibrosis in SSc. These data suggest that CTGF plays a critical role in the development of fibrosis in SSc.

L3 ANSWER 5 OF 14 BIOSIS COPYRIGHT 2002 BIOSIS

2001:299415 Document No.: PREV200100299415. Integrin alphaMbeta2 acts as an adhesion receptor on peripheral blood monocytes and THP-1 cells for Cyr61 and **connective tissue growth factor**
. Schober, Joseph M. (1); Lau, Lester F.; Ye, Richard (1); Ugarova, Tatiana P.; Lam, Stephen C.-T. (1). (1) Pharmacology, University of Illinois, Chicago, IL USA. Blood, (November 16, 2000) Vol. 96, No. 11 Part
1, pp. 18a. print. Meeting Info.: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000 American Society of Hematology. ISSN: 0006-4971. Language: English. Summary Language: English.

AB Cyr61 and **connective tissue growth factor** (CTGF) are growth factor-inducible immediate-early gene products found in normal blood vessel walls, advanced atherosclerotic lesions and healing cutaneous wounds. We previously showed that the adhesion of endothelial cells, platelets and fibroblasts to these matricellular proteins is mediated by integrin receptors. Because monocyte
adhesion is important for inflammation, wound healing and atherosclerosis,
we examined the adhesion of isolated peripheral blood monocytes (PBMC) and
THP-1 cells to microtiter wells coated with Cyr61 or CTGF. Both PBMC and THP-1 cells adhered in a dose-dependent manner to Cyr61- and CTGF-coated wells. Moreover, stimulation of THP-1 cells with 20 muM ADP caused a 6- to
10-fold increase in cell adhesion to both proteins. Time course studies showed that THP-1 cell adhesion to Cyr61 was transient, peaking at 20-30 minutes and declining thereafter. In inhibition studies, while EDTA completely blocked THP-1 cell adhesion to Cyr61, GRGDSP and echistatin had
no effect. These data suggest the involvement of an RDG-insensitive integrin receptor. Using **monoclonal antibodies** specific for integrin subunits, we found that the adhesion of THP-1 cells and PBMC to Cyr61 and CTGF was specifically blocked by YFC118.3 (anti-beta2), and by 44a and 2LPM19c (anti-alphaM). In contrast, mouse IgG
and 6S6 (anti-beta1) had no effect. Thus, monocyte adhesion to Cyr61 and CTGF is mediated by integrin alphaMbeta2. Consistent with the cell adhesion data, a GST-fusion protein containing the I-domain of the alphaM subunit bound specifically to immobilized Cyr61 and CTGF, and the binding was completely blocked by 2LPM19c (anti-alphaM) but not by YFC118.3 (anti-beta2). Collectively, these results identified integrin alphaMbeta2 as an adhesion receptor on monocytes for Cyr61 and CTGF. Since these proteins are synthesized by vascular smooth muscle cells, the interaction of monocytes with these novel extracellular matrix proteins may have an important implication in the physiological function of monocytes.

L3 ANSWER 6 OF 14 CAPLUS COPYRIGHT 2002 ACS

1999:460440 Document No. 131:101260 **Monoclonal antibody** against **connective tissue growth factor** and medicinal uses thereof. Tamatani, Takuya; Tezuka, Katsunari; Sakamoto, Shinji; Takigawa, Masaharu (Japan Tobacco Inc., Japan). PCT Int. Appl. WO 9933878 A1 19990708, 212 pp. DESIGNATED STATES: W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (Japanese). CODEN: PIXXD2. APPLICATION: WO 1998-JP5697 19981216. PRIORITY: JP 1997-367699 19971225; JP 1998-356183 19981215.

AB Disclosed is a human **monoclonal antibody** useful for

remedying various diseases caused by human **connective tissue growth factor** (hCTGF), and preventing the onset of hCTGF-assocd. diseases. Also, disclosed are various **monoclonal antibodies** having various characteristics against various mammalian **connective tissue growth factors** (mCTGFs) useful for detecting and assaying mCTGFs present in the body fluids of mammals suffering from mCTGF-assocd. diseases. The CTGF-assocd. diseases include cell proliferation-accompanying diseases of or fibrosis of lung, hear, liver, kidney, brain, neck, pancreas, stomach, large intestine, small intestine, duodenum, bone marrow, uterus, ovary, testis, prostate gland, skin, mouth, tongue, and blood vessel. Thus, human CTGF (242-252) peptide Cys-Glu-Ala-Asp-Leu-Glu-Glu-Asn-Ile-Lys was synthesized and mixed with Freund's complete adjuvant for immunization to raise polyclonal antibody in rabbits. Similarly, monoclonal anti-hCTGF and mouse CTGF antibodies and producing hybridomas were prepd. Prepd. antibodies were tested for cross binding reactivity and were used for affinity (column) chromatog. purifn. of mammalian CTGFs and treatment of tissue fibrosis in mice model. Mol. cloning of prepd. human monoclonal anti-hCTGF antibody was performed and sequences of single (heavy and light) chain fragments were detd. ELISA with the prepd. antibodies and fragments was used for detecting serum or synovial CTGF in patients with biliary duct obstruction, rheumatoid vasculitis, malignant rheumatoid arthritis, psoriasis, atopic dermatitis, rheumatoid arthritis, osteoarthritis.

L3 ANSWER 7 OF 14 CAPLUS COPYRIGHT 2002 ACS

1999:220010 Document No. 130:251226 Neovascularization inhibitor. Takigawa, Masaharu; Nakanishi, Tohru; Shimo, Tsuyoshi (Japan Tobacco Inc., Japan). PCT Int. Appl. WO 9913910 A1 19990325, 53 pp. DESIGNATED STATES: W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (Japanese). CODEN: PIXXD2. APPLICATION: WO 1998-JP4124 19980911. PRIORITY: JP 1997-267943 19970912; JP 1998-267388 19980904.

AB A medicinal compn. contg. an antibody having a reactivity with human CTGF (**connective tissue growth factor**) has been found out to inhibit the proliferation and migration of vascular endothelial cells and, moreover, neovascularization. It is highly useful in treating diseases and symptoms caused by the proliferation and migration of vascular endothelial cells or neovascularization (for example, diabetic retinopathy, arteriosclerosis, arterial reconstriction, chronic articular rheumatism, psoriasis, sclerema, glaucoma, proliferation or metastasis of tumor, and inflammation in various organs). Thus, recombinant human CTGF was constructed, expressed in HeLa cells, and tested for vascular endothelial cell proliferation and migration-promoting activity. Rabbit monoclonal anti-human CTGF antibody was prepd. using CEADLEEIK as immunogen, and tested for vascular endothelial cell proliferation and migration-inhibiting activity. In addn., transgenic mice were used for raising human monoclonal anti-human CTGF antibodies and hybridomas producing them.

L3 ANSWER 8 OF 14 CAPLUS COPYRIGHT 2002 ACS

1999:130596 Document No. 130:177944 Heparin-binding growth factor (HBGF) polypeptides and their therapeutic uses. Brigstock, David A.; Harding, Paul A. (Childrens Hospital Research Foundation, USA). PCT Int. Appl. WO

9907407 A1 19990218, 53 pp. DESIGNATED STATES: W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1998-US16423 19980806. PRIORITY: US 1997-908526 19970807.

AB Substantially pure heparin-binding growth factor polypeptides (HBGFs), nucleic acids encoding the HBGFs and antibodies which bind to the HBGFs of the invention are provided. These HBGFs are related structurally and functionally to **connective tissue growth factor** (CTGF). The HBGF polypeptides are useful in methods for the induction of bone, cartilage and tissue formation, growth and development of the endometrium and in the acceleration of wound healing. Pharmaceutical compns. contg. the polypeptides of the invention are also claimed.

L3 ANSWER 9 OF 14 MEDLINE DUPLICATE 2

1999377072 Document Number: 99377072. PubMed ID: 10446209.

Activation-dependent adhesion of human platelets to Cyr61 and Fisp12/mouse **connective tissue growth factor** is mediated through integrin alpha(IIb)beta(3). Jedsadayanmata A; Chen C C; Kireeva M L; Lau L F; Lam S C. (Department of Pharmacology, University of Illinois, Chicago, Illinois 60612, USA.) JOURNAL OF BIOLOGICAL CHEMISTRY, (1999 Aug 20) 274 (34) 24321-7. Journal code: HIV; 2985121R. ISSN: 0021-9258. Pub. country: United States. Language: English.

AB Cyr61 and **connective tissue growth factor** (CTGF), members of a newly identified family of extracellular matrix-associated signaling molecules, are found to mediate cell adhesion, promote cell migration and enhance growth factor-induced cell proliferation in vitro, and induce angiogenesis in vivo. We previously showed that vascular endothelial cell adhesion and migration to Cyr61 and Fisp12 (mouse CTGF) are mediated through integrin alpha(v)beta(3). Both Cyr61 and Fisp12/mCTGF are present in normal blood vessel walls, and it has been demonstrated that CTGF is overexpressed in advanced atherosclerotic lesions. In the present study, we examined whether Cyr61 and Fisp12/mCTGF could serve as substrates for platelet adhesion. Agonist (ADP, thrombin, or U46619)-stimulated but not resting platelets adhered to both Cyr61 and Fisp12/mCTGF, and this process was completely inhibited by prostaglandin I(2), which prevents platelet activation. The specificity of Cyr61- and Fisp12/mCTGF-mediated platelet adhesion was demonstrated by specific inhibition of this process with polyclonal anti-Cyr61 and anti-Fisp12/mCTGF antibodies, respectively. The adhesion of ADP-activated platelets to both proteins was divalent cation-dependent and was blocked by RGDS, HHLGGAKQAGDV, or echistatin, but not by RGES. Furthermore, this process was specifically inhibited by the **monoclonal antibody** AP-2 (anti-alpha(IIb)beta(3)), but not by LM609 (anti-alpha(v)beta(3)), indicating that the interaction is mediated through integrin alpha(IIb)beta(3). In a solid phase binding assay, activated alpha(IIb)beta(3), purified by RGD affinity chromatography, bound to immobilized Cyr61 and Fisp12/mCTGF in a dose-dependent and RGD-inhibitable manner. In contrast, unactivated alpha(IIb)beta(3) failed to bind to either protein. Collectively, these findings identify Cyr61 and Fisp12/mCTGF as two novel activation-dependent adhesive ligands for the integrin alpha(IIb)beta(3) on human platelets, and implicate a functional role for these proteins in hemostasis and

thrombosis.

L3 ANSWER 10 OF 14 MEDLINE DUPLICATE 3
1999392959 Document Number: 99392959. PubMed ID: 10465290. Insulin-like growth factor binding protein-4 proteolytic degradation in ovine preovulatory follicles: studies of underlying mechanisms. Mazerbourg S; Zapf J; Bar R S; Brigstock D R; Lalou C; Binoux M; Monget P. (Station INRA de Physiologie de la Reproduction des Mammiferes Domestiques, Nouzilly, France.) ENDOCRINOLOGY, (1999 Sep) 140 (9) 4175-84. Journal code: EGZ; 0375040. ISSN: 0013-7227. Pub. country: United States. Language: English.
AB The regulation of insulin-like growth factor binding protein (IGFBP)-4 proteolytic degradation by insulin-like growth factors (IGFs) has been largely studied in vitro, but not in vivo. The aim of this study was to investigate the involvement of IGFs, IGFBP-2, IGFBP-3, and IGFBP-3 proteolytic fragments in the regulation of IGFBP-4 proteolytic activity in ovine ovarian follicles. Follicular fluid from preovulatory follicles contains proteolytic activity degrading exogenous IGFBP-4. The addition of an excess of IGF-I enhanced IGFBP-4 proteolytic degradation, whereas addition of IGFBP-2 or -3 or **monoclonal antibodies** against IGF-I and -II dose dependently inhibited IGFBP-4 proteolytic degradation. IGF-I and IGF-II, but not LongR3-IGF-I, reversed this inhibition in a dose-dependent manner. C-terminal, but not N-terminal, proteolytic fragments derived from IGFBP-3 (aa 161-264), as well as heparin-binding domain-containing peptides derived from the C-terminal domain of IGFBP-3 and -5 also induced the inhibition of IGFBP-4 proteolytic degradation. Other heparin-binding domain-containing peptides derived from the **connective tissue growth factor** (CTGF) and from proteins not related to IGFBP, heparan/heparin interacting protein (HIP) and vitronectin, but not from p36 subunit of annexin II tetramer, inhibited IGFBP-4 degradation. Furthermore, IGFBP-3, mutated on its heparin-binding domain, was not able to inhibit IGFBP-4 proteolytic degradation. So, in ovine preovulatory follicles, IGFBP-4 proteolytic degradation both 1) depends on IGFs, and 2) is inhibited by IGFBP-3 via its C-terminal heparin-binding domain as well as by heparin-binding domain containing peptides. These data suggest that in early atretic follicles, the increase in IGFBP-2 participates in the decrease in IGFBP-4 degradation. In late atretic follicles, the increase in the levels of C-terminal IGFBP-3 proteolytic fragments, generated by IGFBP-3 degradation, as well as the increase in IGFBP-5 expression would strengthen the inhibition of IGFBP-4 degradation. This inhibition might be partly mediated by direct interaction of IGFBP-4 proteinase(s) and heparin-binding domain within the C-terminal region from IGFBP-3 and -5.

L3 ANSWER 11 OF 14 CAPLUS COPYRIGHT 2002 ACS
1998:708854 Document No. 129:329706 Novel polypeptides within the growth factor superfamily. Holtzman, Douglas (Millennium Biotherapeutics, Inc., USA). PCT Int. Appl. WO 9846641 A1 19981022, 69 pp. DESIGNATED STATES: W: AU, CA, JP; RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE. (English). CODEN: PIXXD2. APPLICATION: WO 1998-US7603 19980415. PRIORITY: US 1997-843651 19970416.
AB The invention relates to Tango-67 polypeptides, nucleic acid mols. encoding Tango-67, and uses thereof. Tango-67 is related to a no. of growth factors, particularly members of the **connective tissue growth factor** family. **Monoclonal antibody** specific for Tango-67 is disclosed for diagnosis or treatment of disorders assocd. with excessive or insufficient expression of Tango-67.

L3 ANSWER 12 OF 14 MEDLINE DUPLICATE 4

1999008896 Document Number: 99008896. PubMed ID: 9790981. Establishment of the enzyme-linked immunosorbent assay for **connective tissue growth factor** (CTGF) and its detection in the sera of biliary atresia. Tamatani T; Kobayashi H; Tezuka K; Sakamoto S; Suzuki K; Nakanishi T; Takigawa M; Miyano T. (Pharmaceutical Frontier Research Laboratories, JT Inc., Yokohama, Kanagawa, 236-0004, Japan.. tamatani@ikrl.jti.co.jp) . BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1998 Oct 29) 251 (3) 748-52. Journal code: 9Y8; 0372516.
ISSN: 0006-291X. Pub. country: United States. Language: English.

AB **Connective tissue growth factor** (CTGF) is a mitogenic, chemotactic, and cell matrix-inducing factor for fibroblasts. We generated murine **monoclonal antibodies** against CTGF and established a sandwich enzyme-linked immunosorbent assay (ELISA) for detection of CTGF. By using the ELISA, we confirmed that CTGF was specifically induced in human fibroblasts by TGF-beta but not by PDGF, FGF, IGF-I, or EGF. We also found that the serum levels of CTGF were significantly correlated with the progression of hepatic fibrosis in biliary atresia. These results indicated that CTGF is potentially a useful parameter for monitoring certain types of fibrotic disorders.

L3 ANSWER 13 OF 14 BIOSIS
1999:459 Document No.: PREV199900000459. Establishment of the enzyme-linked immunosorbent assay for **connective tissue growth factor** (CTGF) and its detection in the sera of biliary atresia. Tamatani, Takuya; Kobayashi, Hiroyuki; Tezuka, Katsunari; Sakamoto, Shinji; Suzuki, Kensuke; Nakanishi, Tohru; Takigawa, Masaharu; Miyano, Takeshi. Pharmaceutical Frontier Res. Lab., JT Inc. 1-13-2 Fukuura, Kanazawa-ku, Yokohama, Kanagawa 236-0004 Japan. Biochemical and Biophysical Research Communications, (Oct. 29, 1998) Vol. 25, No. 3, pp. 748-752. ISSN: 0006-291X. Language: English.

AB **Connective tissue growth factor** (CTGF) is a mitogenic, chemotactic, and cell matrix-inducing factor for fibroblasts. We generated murine **monoclonal antibodies** against CTGF and established a sandwich enzyme-linked immunosorbent assay (ELISA) for detection of CTGF. By using the ELISA, we confirmed that CTGF was specifically induced in human fibroblasts by TGF-beta but not by PDGF, FGF, IGF-I, or EGF. We also found that the serum levels of CTGF were significantly correlated with the progression of hepatic fibrosis in biliary atresia. These results indicated that CTGF is potentially a useful parameter for monitoring certain types of fibrotic disorders.

L3 ANSWER 14 OF 14 CAPLUS
1995:599637 Document No. 123:7874 **Connective tissue growth factor** (CTGF) and antibodies to CTGF.
Grotendorst, Gary R.; Bradham, Jr Douglas M. (University of South Florida, USA). U.S. US 5408040 A 19950418, 12 pp. Cont. of U.S. Ser. No. 752,427, abandoned. (English). CODEN: USXXAM. APPLICATION: US 1993-167628 19931214. PRIORITY: US 1991-752427 19910830.

AB A novel chemotactic and mitogenic protein, **Connective Tissue Growth Factor** (CTGF), a polynucleotide that encodes CTGF and antibodies that bind to CTGF are provided. Diagnostic and therapeutic methods using CTGF are also described. For example, CTGF was partially purified from human umbilical vein endothelial cells with an affinity column contg. immobilized anti-PDGF IgG, the

chemotactic and mitogenic activities of CTGF but not PDGF were identified,
binding of the CTGF to PDGF receptor in endothelial cells was assayed, the
mol. cloning and in vitro transcription and translation of CTGF were described, and the nucleic acid sequence of CTGF was detd. and amino acid sequence was deduced.

=> s l1 and "Hsc24"

L4 0 L1 AND "HSC24"

=> s "FERM BP 6208"

L5 0 "FERM BP 6208"

=> s hybridoma

L6 63348 HYBRIDOMA

=> s l6 and connective tissue growth factor

L7 2 L6 AND CONNECTIVE TISSUE GROWTH FACTOR

=> dup remove l7

PROCESSING COMPLETED FOR L7
L8 2 DUP REMOVE L7 (0 DUPLICATES REMOVED)

=> d l8 1-2 cbib abs

L8 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2002 ACS
1999:460440 Document No. 131:101260 Monoclonal antibody against **connective tissue growth factor** and medicinal uses thereof. Tamatani, Takuya; Tezuka, Katsunari; Sakamoto, Shinji; Takigawa, Masaharu (Japan Tobacco Inc., Japan). PCT Int. Appl. WO 9933878 A1 19990708, 212 pp. DESIGNATED STATES: W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (Japanese). CODEN: PIXXD2. APPLICATION: WO 1998-JP5697 19981216. PRIORITY: JP 1997-367699 19971225; JP 1998-356183 19981215.

AB Disclosed is a human monoclonal antibody useful for remedying various diseases caused by human **connective tissue growth factor** (hCTGF), and preventing the onset of hCTGF-assocd. diseases. Also, disclosed are various monoclonal antibodies having various characteristics against various mammalian **connective tissue growth factors** (mCTGFs) useful for detecting and assaying mCTGFs present in the body fluids of mammals suffering from mCTGF-assocd. diseases. The CTGF-assocd. diseases include cell proliferation-accompanying diseases of or fibrosis of lung, hear, liver, kidney, brain, neck, pancreas, stomach, large intestine, small intestine, duodenum, bone marrow, uterus, ovary, testis, prostate gland, skin, mouth, tongue, and blood vessel. Thus, human CTGF (242-252) peptide Cys-Glu-Ala-Asp-Leu-Glu-Glu-Asn-Ile-Lys was synthesized and mixed with Freund's complete adjuvant for immunization to raise

polyclonal antibody in rabbits. Similarly, monoclonal anti-hCTGF and mouse CTGF antibodies and producing **hybridomas** were prepd. Prepd. antibodies were tested for cross binding reactivity and were used for affinity (column) chromatog. purifn. of mammalian CTGFs and treatment of tissue fibrosis in mice model. Mol. cloning of prepd. human monoclonal anti-hCTGF antibody was performed and sequences of single (heavy and light) chain fragments were detd. ELISA with the prepd. antibodies and fragments was used for detecting serum or synovial CTGF in patients with biliary duct obstruction, rheumatoid vasculitis, malignant rheumatoid arthritis, psoriasis, atopic dermatitis, rheumatoid arthritis, osteoarthritis.

L8 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2002 ACS
1999:220010 Document No. 130:251226 Neovascularization inhibitor. Takigawa, Masaharu; Nakanishi, Tohru; Shimo, Tsuyoshi (Japan Tobacco Inc., Japan). PCT Int. Appl. WO 9913910 A1 19990325, 53 pp. DESIGNATED STATES: W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (Japanese). CODEN: PIXXD2. APPLICATION: WO 1998-JP4124 19980911. PRIORITY: JP 1997-267943 19970912; JP 1998-267388 19980904.

AB A medicinal compn. contg. an antibody having a reactivity with human CTGF (**connective tissue growth factor**) has been found out to inhibit the proliferation and migration of vascular endothelial cells and, moreover, neovascularization. It is highly useful in treating diseases and symptoms caused by the proliferation and migration of vascular endothelial cells or neovascularization (for example, diabetic retinopathy, arteriosclerosis, arterial reconstriction, chronic articular rheumatism, psoriasis, sclerema, glaucoma, proliferation or metastasis of tumor, and inflammation in various organs). Thus, recombinant human CTGF was constructed, expressed in HeLa cells, and tested for vascular endothelial cell proliferation and migration-promoting activity. Rabbit monoclonal anti-human CTGF antibody was prepd. using CEADLEEIK as immunogen, and tested for vascular endothelial cell proliferation and migration-inhibiting activity. In addn., transgenic mice were used for raising human monoclonal anti-human CTGF antibodies and **hybridomas** producing them.

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=> s "FERM BP-6208"

L9            0 "FERM BP-6208"

=> s "FERM BP-6209"

L10           0 "FERM BP-6209"

=> s (tamatani t?/au or tezuka k?/au or sakamoto s?/au or takigawa m?/au)

L11       10250 (TAMATANI T?/AU OR TEZUKA K?/AU OR SAKAMOTO S?/AU OR TAKIGAWA
                M?/AU)

=> s l11 and connective tissue growth factor

L12         139 L11 AND CONNECTIVE TISSUE GROWTH FACTOR
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=> s l12 and monoclonal

L13 8 L12 AND MONOCLONAL

=> dup remove l13

PROCESSING COMPLETED FOR L13
L14 5 DUP REMOVE L13 (3 DUPLICATES REMOVED)

=> d l14 1-5 cbib abs

L14 ANSWER 1 OF 5 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
2000040274 EMBASE Serum levels of **connective tissue growth factor** are elevated in patients with systemic sclerosis: Association with extent of skin sclerosis and severity of pulmonary fibrosis. Sato S.; Nagaoka T.; Hasegawa M.; **Tamatani T.**; Nakanishi T.; **Takigawa M.**; Takehara K.. Dr. S. Sato, Department of Dermatology, Kanazawa Univ. School of Medicine, 13-1 Takaramachi, Kanazawa, Ishikawa 920-8641, Japan. s-sato@med.kanazawa-u.ac.jp. Journal of Rheumatology 27/1 (149-154) 2000.
Refs: 33.
ISSN: 0315-162X. CODEN: JRHUA. Pub. Country: Canada. Language: English. Summary Language: English.
AB Objective. To determine the serum levels and clinical correlation of **connective tissue growth factors** (CTGF) in patients with systemic sclerosis (SSc). Methods. Serum samples from patients with limited cutaneous SSc (lSSc, n = 32), diffuse cutaneous SSc (dSSc, n = 28), systemic lupus erythematosus (SLE, n = 30), polymyositis/dermatomyositis (PM/DM, n = 20), and healthy control subjects (n = 30) were examined by ELISA for detection of CTGF. Results. Serum CTGF levels in patients with SSc were significantly higher than those in patients with SLE or PM/DM, and in controls. CTGF levels in patients with dSSc were significantly higher than those in patients with lSSc. As for clinical correlation of CTGF, SSc patients with elevated CTGF had pulmonary fibrosis, decreased DLCO, and decreased vital capacity more frequently than those with normal CTGF levels. Further, DLCO and vital capacity were inversely and directly correlated with serum CTGF levels in patients with SSc. The dSSc patients with disease duration of 1-3 years had significantly elevated levels of CTGF compared with dSSc patients with duration < 1 year or more than 3 years. Conclusion. Serum CTGF levels were increased in patients with SSc, and correlated with the extent of skin sclerosis and the severity of pulmonary fibrosis. In addition, it appears that production of CTGF is involved in the development or maintenance of fibrosis rather than in initiation of fibrosis in SSc. These data suggest that CTGF plays a critical role in the development of fibrosis in SSc.

L14 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2002 ACS
1999:460440 Document No. 131:101260 **Monoclonal** antibody against **connective tissue growth factor** and medicinal uses thereof. **Tamatani, Takuya; Tezuka, Katsunari; Sakamoto, Shinji; Takigawa, Masaharu** (Japan Tobacco Inc., Japan). PCT Int. Appl. WO 9933878 A1 19990708, 212 pp. DESIGNATED STATES: W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT,

LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (Japanese). CODEN: PIXXD2. APPLICATION: WO 1998-JP5697 19981216. PRIORITY: JP 1997-367699 19971225; JP 1998-356183 19981215.

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L14 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2002 ACS

1999:220010 Document No. 130:251226 Neovascularization inhibitor. **Takigawa, Masaharu**; Nakanishi, Tohru; Shimo, Tsuyoshi (Japan Tobacco Inc., Japan). PCT Int. Appl. WO 9913910 A1 19990325, 53 pp. DESIGNATED STATES: W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (Japanese). CODEN: PIXXD2. APPLICATION: WO 1998-JP4124 19980911. PRIORITY: JP 1997-267943 19970912; JP 1998-267388 19980904.

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L14 ANSWER 4 OF 5 MEDLINE DUPLICATE 1
1999008896 Document Number: 99008896. PubMed ID: 9790981. Establishment of the enzyme-linked immunosorbent assay for **connective tissue growth factor** (CTGF) and its detection in the sera of biliary atresia. **Tamatani T**; Kobayashi H; **Tezuka K**; **Sakamoto S**; Suzuki K; Nakanishi T; **Takigawa M**; Miyano T. (Pharmaceutical Frontier Research Laboratories, JT Inc., Yokohama, Kanagawa, 236-0004, Japan.. tamatani@ikrl.jti.co.jp) . BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1998 Oct 29) 251 (3) 748-52. Journal code: 9Y8; 0372516.
ISSN: 0006-291X. Pub. country: United States. Language: English.
AB **Connective tissue growth factor** (CTGF) is a mitogenic, chemotactic, and cell matrix-inducing factor for fibroblasts. We generated murine **monoclonal** antibodies against CTGF and established a sandwich enzyme-linked immunosorbent assay (ELISA) for detection of CTGF. By using the ELISA, we confirmed that CTGF was specifically induced in human fibroblasts by TGF-beta but not by PDGF, FGF, IGF-I, or EGF. We also found that the serum levels of CTGF were significantly correlated with the progression of hepatic fibrosis in biliary atresia. These results indicated that CTGF is potentially a useful parameter for monitoring certain types of fibrotic disorders.
Copyright 1998 Academic Press.

L14 ANSWER 5 OF 5 BIOSIS COPYRIGHT 2002 BIOSIS
1999:459 Document No.: PREV199900000459. Establishment of the enzyme-linked immunosorbent assay for **connective tissue growth factor** (CTGF) and its detection in the sera of biliary atresia. **Tamatani, Takuya**; Kobayashi, Hiroyuki; **Tezuka, Katsunari**; **Sakamoto, Shinji**; Suzuki, Kensuke; Nakanishi, Tohru; **Takigawa, Masaharu**; Miyano, Takeshi. Pharmaceutical Frontier Res. Lab., JT Inc. 1-13-2 Fukuura, Kanazawa-ku, Yokohama, Kanagawa 236-0004 Japan. Biochemical and Biophysical Research Communications, (Oct. 29, 1998) Vol. 25, No. 3, pp. 748-752. ISSN: 0006-291X. Language: English.
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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	95.28	95.43

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-5.58	-5.58

STN INTERNATIONAL LOGOFF AT 13:40:47 ON 14 JAN 2002